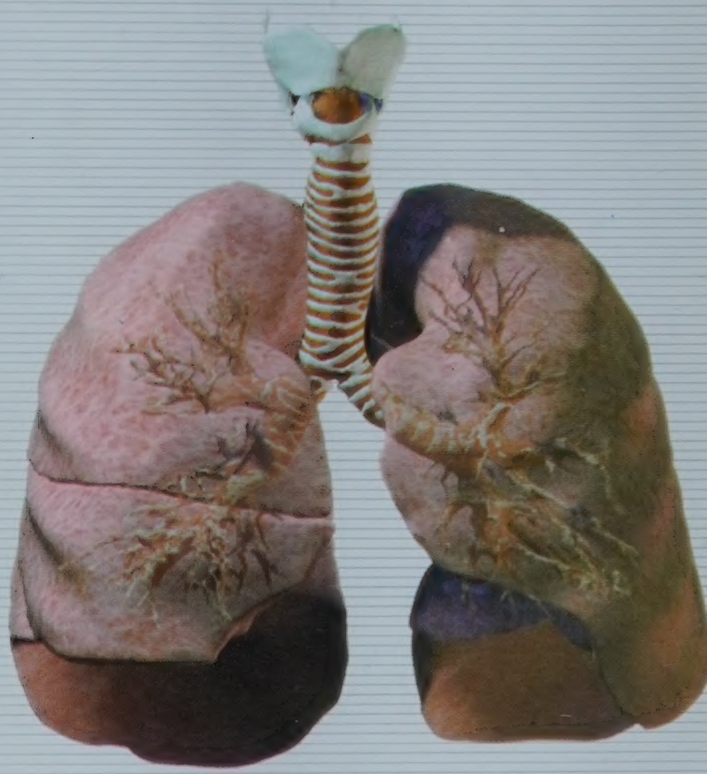
The Fourth Sir Dorabji Tata Symposium

on

Acute Respiratory Disease

10 - 11 March 2003



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In Tropical Diseases
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Department of Microbiology and Cell Biology

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THE FOURTH SIR DORABJI TATA SYMPOSIUM

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March 10 - 11, 2003

Programme & Abstracts

Sir Dorabji Tata Centre for Research
In Tropical Diseases

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Department of Microbiology and Cell Biology
Indian Institute of Science, Bangalore - 560012, India

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FOREWORD

March has come around and it is time for another Sir Dorabji Tata Symposium. We have great pleasure presenting the Fourth Symposium of the series on **Acute Respiratory Disease**. This symposium is timed to coincide with the anniversary of establishment of the Sir Dorabji Tata Trust (11th March).

The topic, chosen by the Scientific Advisory Committee of Sir Dorabji Tata Centre for Research in Tropical Diseases (SDTC) is currently relevant. Acute respiratory infections along with diarrhoeal diseases have been the major killers of young children. Oral rehydration has changed the diarrhoeal diseases scene bringing respiratory infections up to becoming the prime killer of young children in the developing word. Rapid global industralisation has led to the deterioration of the quality of air we breathe, particularly, in the urban conglomerations in the developing countries, sharply brought into focus by the (in) famous Bhopal gas tragedy which is being dealt within this symposium by none other than Dr. S. Sriramachari. This coupled with the spread of tobacco smoking has resulted in an epidemic of respiratory afflictions in the middle and later years of life. This is being increasingly seen in India.

There has been some understanding of the causes of respiratory infections, an improvement in the imaging modalities, a better understanding of the clinical aspects leading to an improved capability of handling infections. Technical advances have improved our capability to support the failing lung. The massive induction of lowlanders during the 1962 Indo-Chinese conflict brought into focus the respiratory problems of living in high altitude. This aspect has been studied extensively by the Armed Forces Medical Services and the Defence Research & Development Organisation. We are grateful to Dr. W. Selvamurthy to agree to talk to us on this subject. The other presentations are by workers in the different aspects of the subject.

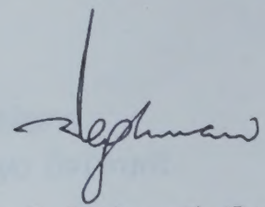
Management of respiratory infections in the community at the basic level would need to be improved if the massive loss of young life is to be ameliorated. We have a satellite symposium on this subject.

In the run up to the symposium it became evident that very little basic research work is being done in this country in the field of Respiration. Even this premier scientific institution has no contribution to make. If this symposium stimulates some research work in this field it would have scored its purpose.

We are extremely fortunate in having Dr. Farokh Udwadia, the doyen of respiratory medicine in India, in our midst. We are sure his keynote address will set the pace of the symposium.

The biology departments of IISc have been very helpful in organising this symposium. They have been generous with their intellectual, organisational and physical inputs.

We wish you a stimulating time and we hope we throw some light on a dark aspect of our health scene.



Lt. Gen. D. Raghunath (Retd.)
Sir Dorabji Tata Centre for
Research in Tropical Diseases

SCIENTIFIC PROGRAMME

Venue:
National Science Seminar Complex (J N Tata Auditorium)
Indian Institute of Science Campus, Bangalore

Monday, March 10 2003

ACUTE RESPIRATORY DISEASE

0900 - 0915	**Inauguration** *Prof. G. Padmanaban, Former Director, IISc, UNESCO Chair, Dept. of Biochemistry, IISc*
0915 - 1000	**Keynote Address** *Dr. F. E. Udwadia, Breach Candy Hospital, Mumbai*
1000 - 1030	TEA

Session I - Clinical Features
Chairperson: Dr. Om Prakash

1030 - 1050	Acute Respiratory Infection Complicating a Road Traffic Accident - A Clinician's Personal Experience *Dr. Sanjiv Rao, Manipal Hospital, Bangalore*
1055 - 1125	Adult Respiratory Distress Syndrome *Dr. Sriram Sampath, St. John's Medical College, Bangalore*
1130 - 1200	Pulmonary Infections in the Tropics *Dr. F. E. Udwadia, Breach Candy Hospital, Mumbai*
1205-1235	Evaluation of Respiratory Distress in Children *Dr. H. Paramesh, Lakeside Medical Research Centre, Bangalore*
1235 - 1300	Discussion
1300 - 1400	Lunch

Session II - Assessment of Pulmonary Functions
Chairpersons: Dr. F. E. Udwadia and Dr. M. R. Sandhya Belwadi

1400 - 1430	Assessing Lung Function *Dr. Om Prakash, St. Martha's Hospital, Bangalore*

SCIENTIFIC PROGRAMME

1435 - 1505	An Overview of Basic Research on Respiratory Physiology with Relevance to Clinical Syndromes in Acute Respiratory Diseases *Dr. K. Ravi, V. P. Chest Institute, New Delhi*
1505- 1535	Pulmonary Imaging in Acute Respiratory Events *Dr. Ravi Kashyap, INMAS, New Delhi*
1540 - 1610	Laboratory Investigation of Acute Respiratory Infections *Wg Cdr (Dr.) P. K. Menon, Command Hospital AF, Bangalore*
1610 - 1625	Discussion
1625 - 1640	TEA

Session III - Therapy of Acute Respiratory Diseases

Chairpersons: AVM P K Sarkar and Dr. Ragini Macaden

1640 - 1710	Mechanical Ventilatory Support for the Failing Lung *Wg Cdr (Dr.) R. M. Sharma, AFMC, Pune*
1715 - 1745	Acute Respiratory Emergencies Associated With Chronic Respiratory Disorders *Col (Dr.) K. E. Rajan, Military Hospital, Pune*

Day 2
Tuesday, March 11 2003

Session IV - Pneumonia and other Acute Respiratory Infections

Chairperson: Dr. Meera D. Meundi

0900 -0930	Acute Bacterial Infections of the Lower Respiratory Tract *Dr. K. N. Brahmadathan, CMC, Vellore*
0935 - 1005	Viral, Mycoplasmal, Chlamydial Infections *Dr. B. Lalitha Rao, NIV, Pune*
1010 - 1040	Role of Molecular Biology in the Rapid Diagnosis of Acute Respiratory Infections *Prof. S. S. Thukral, V.P. Chest Institute, New Delhi*
1040 - 1050	Discussion

SCIENTIFIC PROGRAMME

Session V - Satellite Symposium

1115- 1315 **High Altitude Pulmonary Disease**
Moderator: Dr. W. Selvamurthy, DIPAS, New Delhi

1125-1145 Pathophysiology of Respiratory Maladies at High Altitude
Dr. W. Selvamurthy DIPAS, New Delhi

1145-1205 Role of Nitric Oxide in the Treatment of HAPE
Lt Col (Dr.)B A K Prasad, AFMC, Pune

1205-1225 Indigenisation of Nitric Oxide Therapy for Treatment of HAPE
Lt Col (Dr.) G. Himashree, DIPAS, New Delhi

1225-1245 Lung Surfactant at High Altitude
Dr. Ratan Kumar, DIPAS, New Delhi

1245-1300 Discussion

1300 - 1400 Lunch

Session VI - Environmental Influences on Respiratory System

Chairperson: Prof. R. Nayak

1400-1430 Quality of Air and Respiratory Diseases
Dr. H. R. Rajmohan, Regional Occupational Health Centre (Southern), Bangalore

1430 - 1500 Bhopal Gas Tragedy - An Environmental Disaster
Dr. S. Sriramachari, Institute of Pathology, New Delhi

1500 -1520 Tea

1520 - 1700	**Session VII - Satellite Symposium** **Acute Respiratory Disease in the Community** *Moderator: Dr. Paresh Kumar, CHC, Bangalore*
1530-1600	Community Aspects of Acute Respiratory Infections *Dr. Anuradha Bose, CMC, Vellore*
1600-1630	Socio Economic Aspects of Managing Community Respiratory Infections - Field Experience *Dr. V. S. Sridharan, VGKK, Chamarajanagar*
1630-1700	Public Health Aspects of Acute Respiratory Infections *Dr. Anant Bhan, CHC, Bangalore*
1700-1715	Discussion
1715-1745	Symposium Overview and Valedictory Function
1745	Tea

ABSTRACTS

S-1

Acute Respiratory Infection Complicating a Road Traffic Accident: A Clinician's Personal Experience

Sanjiv Rao
Manipal Hospital, Bangalore

Seldom does one encounter a doctor who has been the treated, with an uncommon, life threatening illness and recovered.

Here a 46 year old practising physician recounts his experience prior to, during his stay in the ICU, and during the recovery.

Particular attention is paid to his emotional experience during his stay in the hospital, what exactly transpired in his mind, and what lasting impression it has made in the person in question on his philosophy of life.

Adult Respiratory Distress Syndrome

Sriram Sampath
SJMCH, Bangalore

Adult/Acute Respiratory Distress Syndrome (ARDS) is a common presentation of acute lung disease in the Intensive care Unit (ICU). It was first described in a group of patients who had hypoxaemia which was refractory to increasing supplements of oxygen, and needed mechanical ventilation (Ashbaugh *et* al1967) This syndrome of tachypnoea, diffuse alveolar infiltrates, and decreased alveolar compliance was termed Adult Respiratory Distress Syndrome to distinguish it from the respiratory distress syndrome seen in neonates (Petty *et al* 1971). An expanded definition was proposed (Murray *et al* 1988), which has been replaced by an easily applicable definition (Bernard *et al* 1994) and ARDS was defined as an Acute Respiratory Distress syndrome since the syndrome was not confined to adults alone.

ARDS can result from a disease process, which sets into motion the systemic inflammatory response. The disease process could be an infectious process like sepsis or a non-infectious process like major trauma or pancreatitis. Local lung disease also can initiate the process and result in ARDS. The changing definitions of ARDS have resulted in the incidence of ARDS varying from 1.5-to 75 cases/100000 population/year. In India an accurate estimate would be difficult due to referral bias and the lack of diagnostic equipment. ARDS usually occurs within 24 hours of the initiating event and nearly always within 3-5 days after development of multi-organ dysfunction. Some of the risk factors associated with a poor outcome in ARDS are chronic liver disease, alcoholism, sepsis, age and active malignancy, while diabetes may be associated with a better outcome due to impaired neutrophil function and lesser lung injury from activated neutrophils. The mortality from ARDS has decreased from around 70% to 40% probably due to improving standards in critical care. The epidemiology of ARDS has been recently reviewed extensively (Atabai 2002)

A diagnosis of ARDS is usually made in an acute setting, when a tachypnoeic patient has bilateral infiltrates on the chest x-ray and arterial blood gas determinations show a PaO2/FiO2 of less than 200, and cardiac failure can be excluded. In ARDS the lung passes from an acute inflammatory phase lasting for a week to a fibrotic phase through a proliferative phase. The acute phase of ARDS is dominated by an exudative process in the alveoli, accompanied by bilateral radiological changes suggestive of a diffuse process similar to pulmonary oedema. This is clinically apparent as hypoxaemia, which requires increasing amounts of oxygen, and the use of positive end expiratory pressure to keep open alveoli which are prone to collapse. Lung function returns to normal in

many patients though some degree of decreased compliance and fibrosis may persist. Activated neutrophils and their products, and products of the inflammatory cascades like cytokines, complement and coagulation pathway mediators affect the alveolus and produce the common clinical picture. The complex interplay of these cytokines has been reviewed (Bellingan GJ 2002, Hotchkiss RS 2003). Mechanical ventilation itself can initiate the process of inflammation in the lung.

It is interesting to note that less than 5% of patients with ARDS die of respiratory failure. The treatment approaches in ARDS have consisted of treating the underlying disease, modifying the nature of the inflammatory response and mechanistic techniques to minimize lung injury. The clinical trials in ARDS have been systematically reviewed (McIntyre *et al* 2000). The presence of multiple disease processes producing a similar pathology in the lung and the genetic heterogeneity of the response to a septic process are confounding factors in ARDS clinical trials. Future directions in ARDS research and treatment would come from a better understanding and quantification of the inflammatory response systemically and in the lung.

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Ashbaugh GD, Bigelow DB, Petty TL, *et al*. Acute Respiratory distress in adults. Lancet 1967, **ii**: 319-323.

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Murray JF, Matthay MA, Luce JM, *et al*. An expanded definition of the adult respiratory distress syndrome. Am Rev Respir Dis 1988, **138**;720-3: erratum 1989; **139**: 1065

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Pulmonary Infections in the Tropics

F. E. Udwadia
Breach Candy Hospital, Mumbai

Pulmonary Infections in the tropics include those seen in the west, and those peculiar to the tropics and developing countries. Tropical pulmonary infections are not just governed geography and environment, but also by socio-economic conditions. Unquestionably, the infection with the most far-reaching consequences in tropical countries is tuberculosis. One third of billion people in India are infected with *Mycobacterium tuberculosis*; around 13 million are infected and diseased and a quarter of these are sputum positive. The increasing incidence of MDR TB and the impact of the HIV epidemic on tuberculosis spells disaster.

Parasites which during their life cycle, migrate through the lungs, are an important cause of non-tuberculous pulmonary infections in the tropics. Infections due to nematodes - ascaris, hookworm, filaria, *Strongyloides* head the list. Parasitic antigens elicit a hypersensitivity response, clinically evident as Pulmonary Eosinophilia, which is characterised, by cough, breathlessness, wheeze, pulmonary infiltrates and a peripheral eosinophilia.

The term "tropical eosinophilia", should be reserved for pulmonary eosinophilia caused by microfilariae of *Wuchereria bancrofti* or *Brugia malayi*. It is invariably associated with an absolute eosinophil count greater than 3,000 per mm^3. and with a good therapeutic response to diethylcarbamazine. Microfilariae trapped within the lungs excite an intense eosinophilic response, leading to eosinophilic pneumonia or bronchopneumonia. Later in the natural history, the cellular exudate within the lungs also includes lymphocytes, monocytes and macrophages with the laying down of fibrous tissue. The end result in untreated cases is a moderate degree of respiratory disability due to patchy pulmonary fibrosis.

Infections with *Strongyloides stercoralis* may be life-threatening in immunocompromised individuals who may develop pneumonia, and lung abscesses from massive invasion of the lungs with filariform larvae.

Echinococcal infection of the lungs results in one or more hydatid cysts within the lung or the mediastinum. A hydatid cyst may rupture into the bronchus, producing an air-fluid level within the cyst, with scoleces in the sputum. Rarely, rupture into the pleural space may cause anaphylaxis.

Schistosomiasis is rare in India, but common in the Middle East. Its pulmonary manifestation include granulomas within the lungs, and pulmonary hypertension with cor pulmonale due to wide-spread blocking of pulmonary vessels with ova of *S. hematobium*.

Protozoal infections of the lung chiefly include *Pl. falciparum* infections and amoebiasis. Pleuropulmonary amoebiasis is invariably secondary to a liver abscess. A raised right dome of the diaphragm with a sympathetic right sided pleural effusion is often observed. Rupture of the liver abscess into the pleura or lung can cause empyema, consolidation and abscess formation in the right lower lobe. A left lobe hepatic abscess can present with similar complications involving the left pleural space and lung. Rupture of a liver abscess into the pericardium is catastrophic event.

Fulminant *Pl. falciparum* infection can cause Acute Lung Injury (ALI), which may be severe (ARDS). Aspiration pneumonia and respiratory muscle fatigue requiring mechanical ventilatory support are also observed. ALI in fulminant *Pl. falciparum* infection is merely one facet of severe multiple organ dysfunction.

Pulmonary toxoplasmosis and pulmonary cryptosporidiosis are protozoal infections seen primarily in immunocompromised individuals.

Pulmonary infections due to *Pseudomonas pseudomellei* (Melidiosis) are chiefly observed in South-East Asia. The acute form of melidiosis is characterized by acute necrotizing pneumonia; the chronic form results in fibrocaseous cavitative upper lobe disease, clinically indistinguishable from pulmonary tuberculosis.

Finally, fulminant bacterial infections like plague, typhoid, anthrax, leptospirosis and other haemorrhagic fevers may involve the lungs, producing pneumonia, pulmonary edema, and acute lung injury.

S-4

Evaluation of Respiratory Distress in Children

H. Paramesh
Lakeside Medical Research Centre, Bangalore

When a clinician encounters a patient with respiratory distress, he should make sure whether the distress is mainly from diseased respiratory system or from other systems like cardiac, CNS, metabolic or musculo skeletal. The clinical evaluation gives more information to identify the site of pathology so that an attempt is made to rectify the physiological derangement while awaiting for specific investigations. Among the five important clinical features like 1. Retractions 2. Stridor 3. Wheeze 4. Grunting and 5. Tachypnea, retractions and inspiratory stridor are cardinal signs in upper airway disease, retractions and wheezing for lower airway obstruction and tachypnea and grunting for paranchymal distress. To establish possible diagnosis, various available tests, their sensitivity, merits and demerits are discussed.

The common causes for respiratory distress in our experience in Bangalore are highlighted.

S-5

Assessing Lung Function

Om Prakash
St. Martha's Hospital, Bangalore

Being exposed to the environment and a variety of other insults, the human lungs are subject to a large number of illnesses. These may primarily affect the airways, the lung parenchyma or the pulmonary vasculature. The clinician has to assess the nature of the illness as well as be able to perform tests that enable quantitation of different aspects of lung function. Commonly performed tests include spirometry, peak flow rate, flow volume loops, test of diffusing capacity and arterial blood gas assay. Special tests such as ventilation-perfusion scan, exercise physiology, bronchoprovocative tests are also availalble, though less commonly done. The commonest test done in clinical practice is spirometry. This is a test of ventilation wherein an electronic spirometer is used to measure forced expiration in terms of the vital capacity and expiratory flow rates. These data, when compared with established norms for the population, allow detection of restricitve, obstructive and mixed types of defects. Spirometry done initially while a particular treatment regimen is employed, allows the clinician to make suitable alterations as needed. Lung function tests are very useful in scientific approach to respiratory illnesses. Meticulous attention, however, has to be paid to the methodology as well as equipment in order to get accurate results. Lung function tests help in diagnosis as well as followup of a variety of diseases affecting the lungs.

An Overview of Basic Research on Respiratory Physiology with Relevance to Clinical Syndromes in Acute Respiratory Diseases

K. Ravi

V. P. Chest Institute, New Delhi

Acute respiratory disease is mainly due to infection of the upper and lower respiratory tract and is caused mostly by viruses, bacteria and fungi. The disease is usually associated with symptoms such as fever, chest pain, cough, tachypnea, airway secretion and dyspnea. Many of these symptoms are considered to be part of airway defence reflexes initiated by sensory receptors of the airways connected to vagal afferents.

The vagal sensory receptors of the airways are classified into: the slowly adapting pulmonary stretch receptors (SARs), the rapidly adapting receptors (RARs), the bronchial C-fiber receptors and the pulmonary C-fiber (type J) receptors. Among these, the former two groups are connected to myelinated vagal afferents and the latter two, to non-myelinated ones. The SARs are responsible for the Hering-Breuer inflation reflex and have mostly a respiration 'regulatory' function. Models of lung disease in common laboratory animals provide experimental evidence that the vagus plays an important role in the airway defence reactions. The tachypnea/apnea and the bronchoconstriction elicited by pulmonary congestion, embolism and anaphylaxis are abolished by vagotomy. Differential vagal cooling demonstrates the involvement of both the RARs and the C-fiber receptors. Neurophysiological studies confirm that in these experimental situations, there is clear activation of these two groups of receptors.

Chemicals that are inhaled or liberated in the bronchial walls stimulate these receptors. The respiratory responses observed in pulmonary embolism have been considered to be due to the liberation of mediators, particularly serotonin, released by the breakdown of platelets. Serotonin activates the C-fiber receptors predominantly. Histamine, bradykinin and prostaglandin F2α - chemicals that are liberated during an acute exacerbation of asthma - excite the RARs and the C-fiber receptors.

Selective activations of the RARs and the C-fiber receptors promote airway secretion, bronchoconstriction and cough. Current evidence indicates that an expansion of the pulmonary extra-vascular space stimulates the RARs preferentially. Following activation of the C-fiber receptors by chemicals, there is the liberation of neuropeptides from these nerve endings (by axon reflex) which will increase the permeability of the microvasculature in the bronchial walls. The fluid that leaks will activate the RARs. Thus, it is likely that the C-fiber induced responses are mediated by the RARs.

These observations in experimental animals suggest that the vagal afferents play an important role in airway defence reactions associated with acute respiratory diseases.

S-7

Pulmonary Imaging in Acute Respiratory Events

Dr. Ravi Kashyap
INMAS, New Delhi

In order to have a comprehensive insight in to pathophysiological changes in acute pulmonary events, it is important to have information regarding biochemical and physiological and anatomical alterations in the cardio-pulmonary system. It is with this knowledge that one can understand the consequences of any acute pulmonary event. The biochemical changes succeed the physiological alterations, which in turn show up as anatomical changes in any disease condition. Imaging modalities provide an insight in to physiological and anatomical changes taking place.

To study physiological and anatomical alterations various imaging modalities are available. Each one complements the information provided by the other, the simplest and the most widely available being x-ray chest examination that allows us to have a two dimensional view of the anatomy of the thorax. Thus allows one to assess to any gross alteration in anatomical details caused by any acute event. X ray chest being simple and portable is a must as the first imaging investigation for assessment of pulmonary pathology. It helps in planning of the acute management or deciding about the future line of imaging technique to be adopted. Ultrasonography is another modality that is portable and widely available. Although normal air filled lung is a bad conductor of ultrasound waves and therefore doesn't show up in a sonographic image but pathological conditions such as solid mass or fluid (pleural effusion, haemothorax etc) can be appreciated by doing sonography. It also helps in guiding diagnostic aspiration and therapeutic intervention through the intercostal spaces. Present day fast Spiral CT examination can evaluate the moving chest with high resolution. The scanning speeds are so fast that it enables effective imaging with short breath hold in an ailing patient. The data is acquired in volumetric mode (3D) and then subsequently projected in 2D or 3D mode. It enables anatomical visualization of both bone and soft tissue (lung, pleura & heart) pathologies by choosing an appropriate window setting on a computer console. Ventilation-Perfusion (VQ) scans allow us to assess physiological alteration in any disease condition. Ventilation and Perfusion scans are performed with radio labeled aerosols (Tc-99m DTPA) and radio labeled MAA (Macro aggregated albumin) respectively. The imaging is performed using gamma camera or SPECT system. A mismatch defect between ventilation and perfusion scan allows us to diagnose pulmonary embolism effectively. This modality also allows us to get a patho-physiological insight in health and disease and can be used to develop and design drugs and drug delivery systems. Magnetic resonance imaging (MRI) with the currently available fast echoplannar systems can effectively image respiratory system, however it has a limited role in acute pulmonary events. It does have a noticeable role in imaging sub acute or cold cases whose acute event has stabilized. Contrast angiography allows us to study the pulmonary vasculature and is particularly useful in diagnosing vascular insults like pulmonary embolism, infarction etc.

To obtain the best from imaging in the management of an acute pulmonary event, it is important to understand the basic principle underlying their utility. X-ray & CT imaging is based on x-ray attenuation ability, ultrasound & doppler is based on echogenicity & ventilation-perfusion scan on patho-physiological alteration in disease of pulmonary system. This helps in delineating and defining the problem and allows for optimal utilization of the various imaging modalities available that can complement the information obtained by biochemical, lung function testing and most importantly a thorough clinical examination of the patient.

Laboratory Investigation of Acute Respiratory Infections

P. K. Menon
CHAF, Bangalore

Respiratory specimens are divided into two major categories, upper **respiratory** and lower **respiratory**. Common upper **respiratory** specimens are throat swabs, nasopharyngeal swabs or washes, and mouth or oral cavity swabs or scrapings. Sinus aspiration and culture for otitis media are submitted only occasionally for specific problematic cases because empirical therapy without culture is generally effective.

From throat swabs, laboratories look for the presence or absence of group A streptococci (*Streptococcus pyogenes*) only unless specifically requested to look for other agents. For other agents of pharyngitis such as *Neisseria gonorrhoeae*, the laboratory must be notified because cultures for agents other than *S. pyogenes* are not routinely done. Throat swabs or nasopharyngeal specimens may also be submitted when diphtheria is suspected, and special smears and isolation media are used for culturing of *C. diphtheriae*. For *Bordetella pertussis* culture, either nasopharyngeal swabs or aspirates are recommended rather than throat swabs or "cough plates."

Lower **respiratory tract infections**, primarily pneumonia, are most often initially cultured from expectorated sputa or tracheal aspirates because these specimens can be obtained with minimal patient discomfort. More aggressive culturing is attained by way of sputum induction for patients producing only scant sputum or by bronchoscopic procedures, including bronchial washes, bronchial brushes, transbronchial biopsies, and bronchoalveolar lavage. In select situations, lung biopsies are performed, particularly when less invasive specimens have yielded no diagnosis. Although sputa and tracheal aspirates are readily obtained, the significance of the potential pathogens that are isolated is sometimes difficult to assess. Also important is that expectorated sputum is insensitive for the detection of *Legionella* or *Pneumocystis*. To eliminate unnecessary as well as potentially misleading culture results, it is imperative to use microscopy screening guidelines to reject sputum specimens that are poor in quality. Rejection is usually based on the observation of many squamous epithelial cells, which generally correlates with significant oropharyngeal contamination. The presence of polymorphonuclear leukocytes usually indicates good specimen quality, but this criterion should not be required in a granulocytopenic patient. Although sputa should be screened when submitted for routine bacterial culture, analogous rejection criteria have not been validated for fungal and mycobacterial cultures.

Lower **respiratory** specimens obtained by bronchoscopic procedures or by lung biopsy should always be regarded as important specimens requiring prompt laboratory attention. Diagnosis obtained by the use of bronchoalveolar lavage and transbronchial biopsy have significantly decreased the need for open lung biopsies. The range of microorganisms that cause lower **respiratory tract infections** is broad, and selection of appropriate tests relies on whether the infection is thought to be community acquired or nosocomial in origin and on other relevant clinical information, including the type of infiltrate, the immunologic status and underlying disease of the patient, history of exposure to known pathogens, travel or occupational exposure, and so on.

In an immunocompromised host the diagnosis of *Pneumocystis* pneumonia can be made from either induced sputum or bronchoalveolar lavage fluid with stains that can be done within a few hours. Calcofluor white stain of wet mount have greatly improved direct detection of fungi. Likewise, the use of fluorochrome smears for acid-fast bacilli rather than the older Ziehl-Neelsen or Kinyoun stains has improved the sensitivity of direct detection of mycobacteria. Radiometric culture methods, as well as other new culture systems for mycobacteria, have reduced time to detection; other advances in mycobacteriology will be discussed. Despite these improvements, more sensitive and specific methods are still needed for detection of *Legionella, Chlamydia, Mycoplasma,* and fungi, particularly *Histoplasma* and *Cryptococcus* in lower **respiratory** specimens. For these and other organisms, molecular amplification assays may be the best direction for improving our diagnostic capabilities

Organism	Acceptable Specimens	Tests Available	Comments
Common bacteria	S, B, L	Gram stain, culture, NAT	1-5 d for isolation and identification
Legionella	B, L	FA,	Poor test, not recommended
		Culture	Requires special media, incubation for 10-12 d
	BL	Nucleic acid amplification	Approved
Mycobacteria	S, IS, B, L	Acid-fast stains	Fluorochrome stains preferred
		Nucleic acid amplification	Approved for smear positive for *M. tuberculosis*
		Culture	Rapid methods preferred, e.g., radiometric 4-6-wk incubation is standard
Nocardia	S, IS, B, L	Modified acid-fast stain	Include positive and negative controls when staining
		Culture	Incubate up to 4 wk

Fungi	S, IS, B, L	Calcofluor white stain	Superior to unstained wet mounts
		Culture	Specify if *Histoplasma* or *Coccidioides* is suspected. Incubate 6 wk
Pneumocystis	IS, B, L	*Pneumocystis* stains stains, etc.	FA, Giemsa, toluidine blue O, silver
M. pneumoniae	S, NP, B, L	No direct stains	
		Culture	Not done routinely, 7-10 d.
	Serum	Serology (CF), Cold agg	4-9-wk conversion time, CA titers nonspecific
C. pneumoniae	NP, ?B, ?L	No direct stains	
		Culture	Fastidious, difficult to grow; not readily available
	Serum	Serology, CF, microimmunofluore scence	Cross reacts within the genus, 4-6-wk conversion time
Viruses	NP, B, L	FA, EIAs,	Available only for certain viruses
		Culture	Rapid shell vials, hold 2-5 d Standard tubes, hold 4-6 wk

Abbreviations:
B, Bronchial washes, brushings, biopsies, bronchoalveolar lavage fluid;
CA, cold agglutinins;
CF, complement fixation;
EIA, enzyme immunoassay;
FA, fluorescent antibody;
IS, induced sputum;
L, lung biopsy;
NP, nasopharyngeal suction or swab;
S, sputum, tracheal aspirates.

*Common bacteria, including staphylococci, streptococci, *Enterobacteriaceae*, *Pseudomonas*, etc.

S-9

Mechanical Ventilatory Support For the Failing Lung

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Respiratory failure is defined as failure of maintenance of normal arterial blood gas tensions. Respiratory failure may be subdivided according to whether the arterial pCO2 is normal or low (type I) or elevated (type Ii). Mechanisms that contribute to respiratory failure include ventilatory failure and venous admixture as a result of either pure intrapulmonary shunt or ventilation/ perfusion mismatch.

Pulmonary diseases which cause severe dysfunction of the mechanics of breathing and requires ventilatory support are ARDS, fulminant pulmonary edema, acute exacerbation of Chronic Obstructive Pulmonary Disease (COPD) and acute severe asthma. The decision to begin mechanical ventilation is a clinical judgment that should take into account the reversibility of the underlying disease process as well as the patients overall medical condition. Parameters that can help to guide the decision as to whether mechanical ventilation is needed include respiratory rate (>35/min), inspiratory force (<25 CmH2O), Vital capacity (<10-15 ml/kg), PaO2 (<60 mm Hgwith FiO2>60%), PaCO2 (>50 mm Hg with PH<7.35), and an absent gag or cough reflex.

When initiating mechanical ventilation certain variables needs to be considered i.e ventilator type,modes of ventilation and setting of ventilatory parameters. Patients on mechanical ventilation should be managed in intensive care unit. Management of a patient on ventilator, ventilatory strategies and weaning modalities in patients with ARDS and acute exacerbation of COPD will be discussed.

S-10

Acute Respiratory Emergencies Associated with Chronic Respiratory Disorders

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Chronic disease processes involving the respiratory system results in a number of alterations in the normal pulmonary physiology and mechanics. Acute Respiratory emergencies occurring in chronically diseased lungs result in altered clinical presentations. A high index of suspicion is essential in these cases to avoid diagnostic pitfalls. Management of such situations necessitates changed strategies. In the current paper, it is envisaged to present a brief discussion on the diagnosis and management of respiratory emergencies in the background of chronically diseased lungs. The following emergencies will be discussed:

1. Infections:
   a) Viral
   b) Bacterial
      (i) Specific
      (ii) Nonspecfic & Anerobic
   (c)Protozoal - PCP

2. Aspiration
   a) Foreign body
   b) Aspiration pneumonias

3. Acute severe asthma
4. Acute respiratory failure & ARDS
5. Pneumothorax
6. Pleurisy, effusion and empyema
7. Pulmonary thromboembolism
8. Goodpasture's Syndrome
9. Emergencies in pulmonary neoplasms including SVC obstruction
10. Miscellaneous
    a) Hemoptysis
    b) Stridor & Wheezing
    c) Sleep related emergencies

S-11

Acute Bacterial Infections Of The Lower Respiratory Tract

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Acute lower respiratory tract infections (ALRTI) are a major cause of morbidity and mortality in children of the developing countries. It is the number one killer disease in children below the age of one year and an estimated one million children below the age of one-year die annually in the developing countries due to ALRTI and its complications. About two-thirds of pneumonias primarily are of viral etiology while the remaining ones are of bacterial etiology. *Streptococcus pneumoniae* and *Haemophilus influenzae* are the predominant causes of primary bacterial etiology while pneumonias of atypical clinical symptomatology are caused by *Chlamydia pneumoniae*, *Mycoplasma pneumoniae* and *Legionella pneumophila*.

An on-going multi-centric study involving six centers located in different parts of India reveal that 13% of the 2178 patients with invasive bacterial diseases were diagnosed as ALRTI. The data from 1999-2002 also show that 121 (42.5%) of 285 confirmed cases of pneumonias were seen in children below the age of one year while only 8.4% of cases were seen in patients of 50 years or more. Fifty-five (19.3%) of the 285 cases of pneumonia were due to *S.pneumoniae* (17.9%) or *H.influenzae* (1.4%). Interestingly, all cases of *H.influenzae* were seen in children below the age of 5 years while 38 (74.5%) of the 51 pneumococcal pneumonias were seen in patients of > 12 years of age.

Antibiotics are routinely recommended for the treatment of ALRTI irrespective of its etiology. This is especially true in countries like India where even viral pneumonias can lead to fatal complications due to secondary bacterial infection. The earlier WHO recommendation to use co-trimoxazole in such cases is no more valid due to the high degree of resistance (54.5%) seen in *S.pneumoniae* and *H.influenzae* to this antimicrobial agent. Penicillin (89%) and erythromycin (96%) continue to be effective drugs because of their high degree of susceptibility.

Although effective vaccines against the above two bacterial agents are available in many countries, they continue to be costly and therefore beyond the reach of people of low socio-economic group in whom ALRTI causes maximum fatality. Effective intervention strategies to minimize risk factors such as low-birth weight, malnutrition, vitamin A deficiency, lack of breast-feeding, indoor pollution and poor sanitation should be considered together with vaccination for the effective control of ALRTI in developing countries.

Acute Respiratory Disease: Viral, Mycoplasmal and Chlamydial Infections

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Acute respiratory disease (ARD) causes enormous morbidity and mortality, particularly in infants and children in developing countries. It is estimated that ARD causes annually over 4 million deaths and millions of episodes of illness in children in developing countries and in India 0.6 million deaths in children under 5 yrs of age. Risk factors that increase the incidence and/or severity of respiratory infections have been identified as malnutrition, crowding and inhaled pollutants all found in lower socio economic classes especially in developing countries. A large number of bacteria and viruses are known to cause ARD with viruses outnumbering bacteria. There are over 200 human respiratory viruses falling mainly within six families, *orthomyxoviruses*, *para myxoviruses*, *picornaviruses*, *coronaviruses*, *adenoviruses* and *herpes viruses*. Many of these respiratory viruses enter the body via respiratory route and cause infection of the respiratory tract and it has been difficult to develop effective vaccines against these viruses. (*influenza*, *parainfluenza*, *corona*, *rhino* etc.). Effective live attenuated vaccines are available for use against some viruses like measles, mumps and rubella which cause respiratory and systemic infections. Bacterial superinfection which commonly occurs following respiratory viral infections and exacerbation of chronic respiratory diseases like asthma during the episodes of respiratory viral infections contribute to morbidity and mortality. Limited antiviral therapy is available for some respiratory viral infections; Amantadine hydrochloride and its analogue rimantadine are antiviral drugs for systemic use in the treatment and prophylaxis of influenza A and ribavirin, administered in a continuous aerosol for 3-6 days is recommended for hospitalized infants with respiratory syncytial virus infection. Rapid diagnostic tests are available for many of these viral infections.

The most important respiratory viruses are *influenza*, *parainfluenza*, *Respiratory Syncytial Virus* (RSV)and *adenovirus*. It is estimated that annually around 0.5 to 1 million die and 600 to 1200 million people become sick due to influenza epidemics worldwide. RSV has been identified as the most important cause of serious lower respiratory tract infection in infants, young children, and also weak elderly people and adults with cardiopulmonary diseases. RSV infection is suspected as possible cause of long term pulmonary dysfunction especially childhood asthma.

Mycoplasma pneumoniae causes respiratory infections ranging from asymptomatic to pneumonia. It generally causes a mild disease. It is a prominent cause of tracheobronchitis and pneumonia in persons of 5-20 years age. The diagnosis of *M. pneumoniae* is mainly done based on clinical

recognition of the illness. Laboratory test is of secondary value. *Chlamydiae* are a group of intracellular organisms and all the three species cause similar respiratory infections but much less frequently than *M. pneumoniae*. Tetracycline and erythromycin are the antibiotics recommended for treating pneumonia caused by mycoplasma and chlamydia.

In India studies on ARD caused by respiratory viruses, mycoplasma and chlamydia are limited. Some investigations conducted in India have shown RSV as an important cause of morbidity in children.

Couple of National Influenza Centres in India have continuously carried out influenza surveillance during the past 50 years. The National Influenza Centres at Coonoor, TamilNadu has investigated and reported the occurrence of pandemics of Asian Influenza A(H2N2) in 1957 and HongKong A(H3N2) in 1968 and other epidemics of influenza regularly during this period. The National Influenza Center at the National Institute of Virology (NIV), Pune, Maharashtra has investigated continuously several outbreaks of influenza over the last 25 years, in Pune. It has been established that influenza outbreaks occur predominantly during rainy season in Pune which has a tropical monsoon climate. It has isolated a total of 384 influenza virus isolates comprising of 42 antigenic variant strains of A(H3N2), A(H1N1) and type B and also bird influenza strains. These include several important global strains that caused extensive epidemics. NIV is associated with World Health Organization (WHO) FLUNET which disseminates update reports on global influenza situation, current vaccines and antivirals.

S-13

Role of Molecular Biology in the Rapid Diagnosis of Acute Respiratory Infections

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Acute respiratory infections are a leading cause of morbidity and mortality worldwide, accounting for about 30% of all childhood deaths in the developing world. The causative agents of ARIs encompass a wide variety of microorganisms. Molecular techniques provide for the sensitive and specific detection of putative pathogens. In general, molecular techniques are indicated for the detection of organisms that cannot be grown in vitro or for the detection of organisms requiring complex media/ cell culture. A large variety of respiratory pathogens viz: respiratory viruses (adenoviruses, influenza, parainfluenza, RSV, Rhinoviruses,coronaviruses and enteroviruses etc.), and bacteria (*Bordetella pertusis, Legionella spp.. Chlamydia spp., Mycoplasma pneumoniae)* meet the above mentioned criteria. The use of specific nucleic acid probes was shown to have a limitation in its lack of sensitivity. Currently available nucleic acid amplification techniques such as PCR and reverse transcription, RT-PCR are highly sensitive techniques for the detection of nucleic acid sequences from viruses and bacteria in clinical samples. However, the diagnostic utility of nucleic acid amplification techniques for a single infectious agent is limited by the inability to establish a specific etiology. The awareness of this limitation led to development of multiplex PCR assays and multiplex RT-PCR assays for simultaneous detection of pathogens. Recent attempts have been directed at using a multiplex RT-PCR (m-RT-PCR) protocol which allows simultaneous detection of a variety of respiratory viruses and bacteria from clinical specimens. These studies are expected to yield overall benefits viz: rapid diagnosis, rational use of antibiotics, fewer side effects, reduction in the emergence of antibiotic resistance, reduced hospitalization rate and duration and reduced requirements for diagnostic procedures.

S-14

Pathophysiology of Respiratory maladies at High Altitude

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Respiratory compensation is a prompt and significant response in the process of acclimatization at High Altitude (HA). In the people who maladapt at HA the two respiratory maladies i.e. pulmonary edema (HAPE) and Pulmonary artery hypertension (PAH) may occur. Hypoxic stress at HA stimulates hyper ventialtion through the chemoreceptors, which is an import adaptive feature. It also leads to increase in pulmonary artery pressure through the stimulation of alpha sympathetic receptors in the pulmonary vascular bed. The sympathetic mediated peripheral and splanchnic vasoconstriction leads to increase in the load on the pulmonary vascular bed. There is also increase in the pulmonary capillary permeability and decrease in the surfactant activity. Due to this, small molecular proteins like albumin leak into interstitial space drawing more water due to elevated osmotic pressure. J-Receptor stimulation at this juncture will try to offer some physiological protection. However, if the individual consciously suppresses it at the central level and continues to exert physically or emotionally apprehensive about the new environment, it may lead to the development of HAPE. Hypothalamus and sympthetic nervous system play a pivotal role in modulating these responses. Defence R & D Organisation (DRDO) has developed Hyperbaric chamber for treatment of HAPE cases and also a new treatment modality using the inhalation of Nitric Oxide (25 ppm) and oxygen (50%). A new aerosol administration of pulmonary vasodilators using a modified Nebuliser has also been successfully devised. These research accomplishments will be discussed.

S-15

Role of Nitric Oxide in the Treatment of High Altitude Pulmonary Edema

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High Altitude Pulmonary Edema (HAPE) is a life threatening condition seen on ascent to high altitude, characterised by hypoxemia, pulmonary hypertension and increased pulmonary capillary permeability. Lowering pulmonary arterial pressures by vasodilators shows improvement in symptoms suggesting pulmonary vasoconstriction has a role in HAPE. Acute effects of inhaled Nitric Oxide (NO), Oxygen and mixture of Oxygen plus NO on HAPE patients has shown that NO plus oxygen has additive effects on pulmonary hemodynamics and even greater effects on gas exchange, indicating a therapeutic role of NO in HAPE.

Results of the clinical studies carried out in the author's laboratory with HAPE patients and controls with different treatment regimens indicate that NO with Oxygen has a strong therapeutic role in the management of HAPE.

S-16

Indigenisation of Nitric Oxide Therapy for Treatment of High Altitude Pulmonary Edema

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In the respiratory tract Nitric Oxide (NO) plays an important signaling role in the physiological control of airway function and in the patho-physiology of airway diseases. High altitude pulmonary edema (HAPE) is a life threatening condition, which occurs on rapid ascent to altitudes higher than 2500m. The main pathology in this disease is hypoxia induced pulmonary vasoconstriction followed by hydrostatic imbalance and edema. Any therapy, which achieves pulmonary vasodilatation, is sure to cause reversal of pulmonary edema and improvement in symptoms. Another advantage of NO is that it does not produce any change in systemic arterial pressure or systemic vascular resistance making NO the first seemingly selective pulmonary vasodilator. Inhaled Nitric Oxide (INO) with oxygen has shown to be of immense therapeutic value in patients of HAPE. NO has found a place in treatment of multitude of diseases. The current applications of INO include Hypoxic respiratory failure in neonates, pediatric pulmonary hypertension, acute respiratory distress syndrome in all age groups, primary pulmonary hypertension, cardiac transplants, lung transplants, post surgery right ventricular dysfunction in congenital heart disease, and HAPE. The commercially available INO machines are not suitable for patients of HAPE, as these delivery systems are ventilator based. Patients of HAPE require a machine that can deliver NO without a ventilator. We have designed a simple and easy to use NO delivery system for spontaneously breathing patients and this holds great promise in treatment of HAPE.

Key words: Inhaled nitric oxide, High altitude pulmonary edema, Nitric oxide delivery systems

Lung Surfactant at High Altitude

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The sojourners exposed to adverse climatic conditions of high altitude (HA) suffer from various HA induced diseases. High altitude pulmonary edema (HAPE) is one of the most dreaded, and is often fatal unless prompt action is taken to deport the patient to lower altitudes or given supplemental oxygen therapy. Many hypotheses have been advanced to explain the aetio-pathogenesis of HAPE but none of them have been accepted, suggesting it to be a multi-factorial disease.

Role of lung surfactant in development of HAPE has been suggested. Lung surfactant is a lipo-protein, and present as an alveolar lining between the air and alveolar surface. Phosphatidyl choline (lecithin) is the most abundant and surface-active alveolar lipid present in lung surfactant and it showed good correlation between amount of pulmonary lecithin and alveolar tendency to collapse i.e. atelectasis. The protein part of lung surfactant consists of four surfactant proteins (SPs), SP-A, SP-B, SP-C and SP-D. SP-A is a major surfactant protein that plays important role in regulating the surfactant synthesis, spreading on the alveolar surface and re-utilization of its components. Lung surfactant serves three important functions, i) lowers surface tension adjusted to varying alveolar radius, preventing atelectasis ii) together with tissue forces and plasma osmotic pressure it resists the effusion of plasma into alveolar spaces thereby, preventing development of pulmonary edema and iii) it provides pulmonary defense response from small particles through alveolar macrophages.

Most of the lung surfactant studies in relation to HAPE are limited to animal studies only as it is difficult to obtain broncho-alveolar lavage (BAL) from human subjects when they suffered from HAPE. Simulated acute (7620 m, 5 hrs.) and chronic (6096 m, 21 days) hypoxia produced pulmonary edema in rats. Acute hypoxia of 7620 m lowered phospholipid, specifically phosphatidyl choline, most surface active component, in rat BAL. But superimposition of cold (5°C) on hypoxia further lowered phospholipids both in BAL and lung tissue. In chronic hypoxia phospholipids content of lung tissue and BAL both decreased. Superimposition of cold (5°C) caused greater decrease as compared to chronic hypoxia alone. This suggested that in development of HAPE in animal model, effect of hypoxia alone in lowering surfactant is related to the duration of exposure and superimposition of cold on hypoxia becomes more damaging.

Environmental and genetic interactions play a vital role in adaptation during exposure to adverse situations. In patients suffering from HAPE there is possibility of gene mutation/polymorphism in SPs, which play important role in functioning of lung surfactant. In human subjects suffering from HAPE, both sojourners and natives, SP-A gene polymorphism has been studied in small number of subjects.

Quality of Air And Respiratory Diseases

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Quality of air is extremely important from the point of hazard free respirable air which is essential for breathing. Normally the air, constituent of environment in which we live, has a built-in mechanism to purify itself. The air gets polluted due to the continuous interaction of atmosphere, lithosphere, hydrosphere and biosphere resulting in complex multifarious hazardous situations in ambient environment. Circumstances are almost similar in case of work room/occupational environment, also due to processes.

Environment problem started getting considerable attention after 1972, through the Stockholm conference. Growth of civilisation through various activities is responsible for the environment related health problems. There has been a shift in the disease profile away from infectious diseases to chronic diseases as a cause of suffering disability and death.

Lungs as portals of entry with potential direct or reflex effects on the organ systems, the relevance of quality of air and respiratory diseases attracts major attention. As early as 1971 when natural air quality standards were first established by US. Environmental Protection agency over 100 million citizens were identified to be exposed to environmental irritants from primary air pollutants. Now the fourth leading cause of death being pulmonary diseases, with 20 million Americans exposed to potential respiratory hazards in the work place. As we do not have authentic documents to quote the burden of such acute and chronic respiratory diseases it is necessary to recognise the association with quality of air, environmental and occupational exposures. Little has been done in the last few decades to reduce the burden of these respiratory conditions at the community level.

The presentation is focused to deliberate broader areas of understanding of exposure, acute and chronic respiratory diseases, patho physiologic mechanism of disease development and prospective epidemiological studies. Hypothesis and interactions between susceptibility and environmental occupational exposure need to be explored in terms of gene environment, host interaction that are altered by growth of the developing lung, aging of the lungs, changes in defense mechanisms resulting from smoking, infection, dietary and nutritional factors, influence of man made environment and strategies for the abatement of such exposures to reduce the burden of respiratory and other systemic diseases.

S-19

Bhopal Gas Tragedy - An Environmental Disaster

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Even after Eighteen years, the December 1984 Bhopal Gas Tragedy following the leak of 42 tons of Methyl Isocyanate (MIC) from the UCC plant, is unparalleled in the history of chemical disasters in the world. In its aftermath, over 3000 people died; several remained maimed and crippled for life. The State Health Authorities ably faced the challenge, with prompt and unstinted support of the ICMR. Over 25 research projects were sanctioned, practically on every conceivable aspect of the human tragedy. This presentation is intended to focus on the results of two studies related to the Pathology & Toxicology and Patho-physiology following inhalation of MIC along with several aqueous and pyrolytic decomposition products which were released into the Environment.

Human Autopsies: Within hours of the deadly leak, hundreds perished and scores of dead bodies started pouring into the morgue. Late Prof. Heeresh Chandra and his dedicated team at the Medico Legal Institute, Bhopal, subjected them to meticulous Post-Mortems. He described vividly the **"Cherry Red"** discoloration of the blood and lungs, in particular. This unique finding set the pace for a multi-pronged Toxicological investigation. Indeed, I was fortunate to participate actively in these studies right from the inception. My colleagues, Dr. H.M.K.Saxena and Dr. Ashok Mukherjee & Dr. A.K.Jain from the Institute of Pathology also joined the team to help us in Light & Electron microscopic studies.

Histopathology: Literally, the hundreds of Autopsies were categorized into Acute, Sub-acute and Chronic phases. Dysponea at rest, accentuated on exertion characterized the "Acute phase" and was accompanied universally by ocular involvement and cough. Clinically, the situation was comparable to ARDS (Acute Respiratory Distress Syndrome). Yet another striking feature was extreme muscular weakness. Autopsies in this early phase revealed generalized edema and marked congestion of the viscera, especially of the respiratory tract and the brain. The lung, which was the most damaged organ, weighed over twice the normal. Microscopically, there was widespread intra-alveolar & interstitial transudation of albuminous fluid and emphysematous bullae. Inflammatory changes were minimal. In fact, at that early stage up to January 1985, while the hazard of severe hypoxia was recognized, even some Indian scientists envisaged a good prognosis for the Bhopal survivors, on the analogy of High Altitude Pulmonary Oedema (HAPO)! It was even doubted whether the severe pulmonary edema caused by the chemical MIC could really lead to any permanent tissue damage.

But these hopes were soon belied by the findings in the subsequent **"Sub-acute phase"**. The lungs showed progressive exudative changes, with varying degrees of bronchitis, bronchiolitis and widespread pneumonitis. Still later, in the **"Chronic phase"**, there was evidence of organization and consolidation, with widespread diffuse interstitial pneumonitis and fibrosis **(DIPF & GIPF)**, clinically corresponding to **COPD**. While a small group had persistent damage, surprisingly enough, a large proportion of people slowly recovered.

The brain was edematous to the naked eye and had extensive hemorrhages, both grossly and microscopically. Ring hemorrhages and Pericellular and Pericapillary edema were observed under Light & Electron microscopy. All these features were suggestive of acute anoxic or histo-toxic damage.

Experimental Studies: Dr. K Jeevaratnam of DRDE, Gwalior and myself successfully developed a rat model. Following single exposures of MIC or its derivatives, methyl Amine (MA) & Dimethyl Urea (DMU), over a period of 10weeks, the entire spectrum of lesions, from pulmonary edema to fibrosis, were reproduced.

Issue of Cyanide toxicity: As already stated, a glaring feature of the pulmonary damage was the "Cherry Red" discoloration of the lung as well as the contained blood. Traditional and newer causes for the same were explored in a systematic manner.

(a) The possibility of Carbon Monoxide as the primary, though transient, cause was ruled out after preliminary spectroscopic examination.

(b) Hence, Prof. Chandra strongly invoked a possible exposure to HCN (Hydrogen Cyanide). This resulted in a needless major controversy, in spite of a recent paper by Blake & Masjudi. In fact, subsequent analysis of preserved blood samples of Bhopal victims showed elevated levels of cyanide.

(c) The prompt therapeutic response to NaTS (Sodium Thiosulphate), substantiated the hypothesis, which was further confirmed by elevated levels of urinary SCN (Thiocyanate), in individual cases and in "Controlled Double Blind Trials, before advocating its wider use.

(d) Interestingly enough, the marked muscle weakness dramatically improved indicating pari passu involvement of Myoglobin and/or circulatory disturbances.

(e) Concurrently, relevant **patho-physiological studies** were carried out. Firstly, for over a year, there was marked and sustained elevation of Hb (Haemoglobin). Secondly, Late Prof. P.S.Narayanan demonstrated significant alterations in the Blood Gases of 'Acute'

cases. Both Arterial and Venous Oxygen (O2) were lowered, surprisingly alongside lowered Arterial/Venous levels of CO2 (Carbondioxide), which did not increase after exercise. However, NaTS therapy was accompanied by substantial improvement following exercise, in the Oxygen carrying capacity as well as Venous CO2, both in the Peripheral & Central Venous Pool.

(f) The levels of 2 -3 DPG (DiPhosphoGlycerate) levels were estimated with the help of Shri. K. Sridharan & Dr. Patil of DIPAS. Initially, the values were found to be elevated to more than twice the normal, as if the patient had been exposed to an environment of HA (High Altitudes) of about 14,000 feet.

(g) However all the aforesaid physiological alterations of the blood caused by MIC exposure, tended to return to normal with the passage of time.

(h) However, one disturbing fact was the episodes of periodic recurrence of respiratory symptoms, which often responded to repeat NaTS therapy with simultaneous elevated urinary SCN. Such a phenomenon could not be accounted for in the absence of repeated-exposures to HCN.

Carbamoylation: As reported in the literature and urged by Dr. Sriramachari from the beginning, there was a need to explore possible direct binding of MIC to Hb and tissue proteins,. In view of the phenomenon of recurrence, this line was pursued more vigorously. With the help of Prof. Ramaiah of AIIMS and later the DRDE team under Dr. P.K Ramachandran, the binding of MIC to the end-terminal Valine residues of the Hemoglobin chains was first established. The consequent failure to pick up CO2, which in turn impaired the release of Oxygen (O2), finally seemed to explain the enigma of the **"Cherry Red Discoloration"**. Extended studies on deceased victims and survivors in the Bhopal Projects fully confirmed that MIC binds, not only to Hb, but several other end-terminal amino acids of tissue proteins. Interestingly, it was found in the initial phases that several 'Erythrocytes appeared Electron-lucent under EM, suggestive of leaching out of some substance possibly S-Carbamoylated Glutothione.

From the literature it is well known that, unlike irreversible N-Carbamoylation, the binding of MIC to Sulphydryl groups is not only fast but **reversible** and could act as 'depots' for 'ferrying around' the MIC molecule. Thus, the underlying causes of reversible muscular weakness and the protracted excretion of thiocyanate might have been elucidated. Indeed, Baillie and Slatter confirmed such a possibility with the help of the sophisticated C.I.-M.S. technique. Unfortunately, due to want of appropriate equipment we could not fulfill our dream of demonstration of S-Carbamoylation of not only Glutathione but also Sulfhydryl containing enzymes like Rhodanese,

Cholinesterase and Aldolase of muscle. The underlying causes of reversible muscular weakness and the protracted excretion of thiocyanate might explain the unusual type **chronic cyanide** toxicity. It was unfortunate that due to lack of access to appropriate equipment, we could not fulfill our original dream of establishing the

"Biochemical Lesion of Bhopal Gas Tragedy". It would also appear that the Environmental Disaster of Bhopal was unique in that the "MIC was not cold but pyrolysed" and contained, apart from HCN, more reactive "chemical adducts".

Summing up, the Pulmonary and Cerebral Edema and the Sequelae of the Bhopal Tragedy, are manifestations of severe **"Histo-toxic Anoxia"** aggravated by metabolic disturbances following **N- & S- Carbamoylation.**

An attempt will be made to portray briefly the salient features of some of the facets of the pathological and toxicological findings.

S-20

Community Aspects of Acute Respiratory Infections

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Acute lower respiratory infection is a leading cause of morbidity and mortality in children less than 5 years of age. The proportion of deaths directly attributable to ARI is 23% in under-5s. Pneumonia causes 4 million deaths world-wide, 2/3 of which occur in developing countries.

Magnitude of the problem:

Acute Respiratory Infections is one of the two leading causes of admissions into our hospital in the paediatric age group. The incidence of ARI is 8-9 episodes per child per year in the first 2 years of life, and drops to 3-4 episodes in the next 2 years. Hospital data may not reflect true community incidence. A more accurate estimate would be got by doing community surveillance supplemented by verbal autopsies of all deaths in this age group.

ARI: Etiology

The most common paediatric pulmonary infection is non-bacterial. Below 5 years of age, the commonest cause is *Respiratory Syncytial Virus* and above that age group it is *M.pneumoniae*. In developing countries, bacterial pathogens are thought to play a greater role in severe ARI than in developed countries.

Diagnosis of ARI:

The WHO criteria for the diagnosis of Acute Respiratory Infections and the guidelines in the modules of the Integrated Management of Childhood Illnesses are useful in the training of young physicians who do not have the expertise of paediatricians but who nevertheless have to deal with paediatric patients as part of their work in teaching hospitals and in smaller hospitals.

The definitions of ARI are clear:

Severe pneumonia: Four criteria: 1) tachypnoea; (respiratory rate >50 / min); 2) any one of the **danger signs**: chest indrawing, central cyanosis, inability to feed/ lethargy; 3) Fever (temperature > 38 C), 4) Crepitations on auscultation. Infants with concomitant wheezing will fit the definition. Wheezing without the other abnormal auscultatory findings will not fit the definition.

Clinical guidelines on those children who would benefit from hospitalization would be helpful in maximally utilizing available resources in developing countries. Several clinical aspects may be used in assessing need for hospitalization (i.e. young age, malnutrition, underlying chronic illness, tachypnoea, chest indrawing and somnolence) for children with pneumonia.

Link between illness and malnutrition:

Seven out of ten deaths among the world's children are caused by infectious diseases. Malnutrition is a contributing cause in more than half of the children's deaths. At present, interventions against such diseases in children are the most cost-effective way of reducing the world's morbidity and mortality.

Cellular immunity has been linked to micronutrient deficiency. The micronutrients which have been studied extensively are Vitamin A and recently, zinc. Recent studies have shown that zinc given during an acute episode of ARI, is effective in reducing the duration and severity of pneumonia. Whether this would be applicable as a public health strategy is not clear, and more work needs to be done in this field.

Treatment:

The WHO recommended strategy for control of mortality due to ARI is to use a case-management approach and to give antibiotics to those children where a diagnosis of ARI has been made based on the definitions of ARI (see above).

Can patients be treated as out-patients or do they require admission?

It is possible that admission could be restricted to children who exhibit the danger signs of pneumonia and also those children who have the additional risk factors of young age(<2 years), underlying chronic disease or malnutrition.

Prevention:

The prevention of ARI requires a move from an individual oriented strategy to a population oriented one that seeks to control the determinants of disease in the population. Infections are intricately linked to malnutrition, poverty and ignorance. Mortality in ARI is determined by delay in seeking health care, malnutrition, poverty and gender bias.

What is required then is:

1) Community based research to study the incidence and prevalence of common respiratory illnesses.
2) To increase awareness of the case-management approach among public and private physicians.
3) Research on the value of micro-nutrient supplements to reduce morbidity and mortality of respiratory infections.
4) To increase awareness about the early recognition of illness in young children and recognition of danger signs of the disease which would necessitate referral to a health care facility.
5) Nutritional rehabilitation and diminution of the problem of malnutrition in our country.
6) Improvement in the social status of women.

S-21

Socio Economic Aspects of Managing Community Respiratory infections: Field Experience

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This paper is the outcome of field experience at both the Tribal area and the Non tribal impoverished rural area.

The socio economic status has its impact on the following aspects of any illness, more so respiratory infections:

- Aetiology
- Diagnosis
- Health seeking behavior of the people concerned
- Availability of resources
  - Health manpower
  - Money and materials
  - Time
- Prevention of the illness
- Rehabilitation

Some of the special situations complicating the already strained economy will also be mentioned, namely,Sickle cell disease in the tribal area, Diabetes mellitus, Post tubercular pulmonary damage predisposing to secondary infections, HIV/AIDS.

The role of using simple devices like the peak flow meters and nebulizers in providing optimal care and the role of inhalers in asthma in an economically deprived society will be outlined.

The field experience shows many modifications and compromises of the conventional methods of treatment of respiratory infections and also brings out the lacuna in what is taught in the medical colleges and what is possible in the community. There is an urgent need to address this as the **future seems to be bright for the Microbes !**

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Public Health Aspects of Acute Respiratory Infections

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Infections of the respiratory tract are the most common human ailment. While they are a source of discomfort, disability and loss of time for adults, they are a substantial cause of morbidity and mortality in young children and the elderly. In India, ARIs are one of the major causes of death in states and districts with high infant and child morality rate.

The poster will attempt to examine the public health aspects of Acute Respiratory Infections and the various factors involved such as environment (including housing, industrialization), nutrition (including breast feeding), low birth weight, Vitamin A deficiency and other host factors, poverty, overcrowding, poor ventilation, unclean surroundings, occupational factors and lack of awareness etc. The SEPC (Social, Economic, Political and Cultural) Analysis of the causative factors will help in understanding the associated problems.

The importance of locating action for prevention and control of Acute Respiratory Infections in a comprehensive health care context by strengthening primary health care services to provide better Maternal & Child Health facilities, universal immunization, improved nutrition and decreasing indoor smoke pollution will be highlighted.

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